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Sickle Cell Anemia in Pregnancy

HYMAN E. CANTER, B.Sc., M.D., M.Sc. (MED.), F.I.C.S.

Anemia in pregnancy is not an uncommon occurrence. It has been estimated that about 25% of all obstetrical patients will show a moderate grade of anemia, especially in the last trimester of pregnancy. This anemia is not due to the hydremia which causes dilution of the red blood corpuscles, but there is an actual diminution in the amount of hemoglobin and a decrease in the number of red blood corpuscles. Many pregnant women will show a hemoglobin of less than 12 Gm. and red count of less than 4 million. However, the type of anemia we are about to describe is an unusual one. First, in its comparative rarity, and second, in its predilection for the Negro race.

Sickle cell anemia was first discovered by Dresbah in 1904, but it was not until 1910 that Herrick[1] described this disease and differentiated between the acute phase, that is the anemic phase, to which the term Sickle Cell Anemia was given, and the asymptomatic sickle cell trait in which the name Sicklemia was used. In sickle cell anemia there is not only sickling of the red blood cells, but there is also evidence of active destruction of the cells as shown by increased regeneration, such as reticulocytosis and nucleated red cells (normoblasts). Destruction is shown also by increased icterus index, bilirubinemia and jaundice. In sicklemia the red cells are normal in number but are abnormally shaped, and there is no evidence of blood destruction. The ratio of sickle cell anemia to sicklemia is about 1:40.

Sickle cell anemia is a hemolytic disease found almost exclusively in the Negro race, although a few reports have cast some doubt as to whether or not it can occur in other races. Killingsworth and Wallace[2] found one case of this disease in a Mexican and Fowler has seen a case in the white race. Both sicklemia and sickle cell anemia are transmitted to either sex as a dominant Mendelian factor. About 8% of the Negro race shows the sickling trait of the red blood cells. One out of 15 cases according to Wintrobe[3] and Kracke[4] develop true sickle cell anemia. The degree of anemia in this disease varies considerably from case to case and from time to time in the same patient. During periods of exacerbations, it may be very severe, the red cell count falling to a level of 1 million with a proportionate reduction in hemoglobin. In addition to the sickle cells, there will be found round cells with central and peripheral dark areas separated by a pale zone. These "target cells" are frequent in this disease. Nucleated red cells, normoblasts, are common and the reticulocytes are increased. Leukocytosis is also common.

The majority of these patients are very young. The disease manifests itself early in life, about the first two decades. Many of them die before they reach puberty and those who reach maturity will generally die before the age of thirty. They are usually unable to become pregnant. The infertility may be due to hypoplasia of the genital organs and the severe anemia which may suppress ovulation.

Pregnancy seems to have a very deleterious effect on the mother and the fetus. Many of these patients will abort and some of those who do carry a longer period will give birth to prematures, macerated fetus or stillborns. Toxemia is also more frequent than in the normal pregnant Negro woman, as are infections, especially pulmonary and puerperal sepsis. Postpartum hemorrhage is not an uncommon occurrence.

Up to this time, there are only 27 cases of pregnancy with sickle cell anemia reported in the medical literature. The first case was reported in 1924 by Sydenstricker[5]. In 1931 Yader[6] reported 2 cases, Lash[7] in 1934 and Richter[8] in 1935 and Sharp and Schleiter[9] in 1936 and Killingsworth and Wallace[2] in the same year each reported one case. Lewis[10] in 1937; Sodeman and Burch[11] in 1938; and Page and Silton[12] in 1939; each reported a case. Then in 1949 Kobak[13] and his associates added six additional cases of their own. In 1947 Martinek[16] reported 2 cases. Karangelo and Otts[17] reported six cases making a total of 27. Of this number there were seven maternal deaths, a mortality rate of 26%. There were 57 pregnancies among 27 cases, and of these 18 were primiparas and 9 were multigravida. Two-thirds of these births were females and one-third males. The infant mortality was 52%. There were only three patients with this disease in pregnancy who were past the age of thirty. The oldest patient was a 35-year-old multipara reported by Spivak in 1945. The rarity of this disease in pregnancy justifies the reporting of an additional case.

Case History

J. W. G., a twenty-year-old negro, gravida I, para O, approximately seven months' pregnant, was admitted to the Montefiore Hospital on May 24, 1948, because of severe abdominal pain; headaches, pains in the shoulder, elbow, knee joints, and stiffness of the neck. She had had occipitofrontal headaches at various intervals for the past two months, but they had become more severe that evening. The pain in the abdomen radiated down both legs. There was nausea and some vomiting.

Past Medical History: Patient had rheumatic fever in 1942; jaundice lasting for 3 weeks in 1944; and lobar pneumonia in 1947.

Menstrual History: She reached puberty at age of 13; periods were regular, 28 days of 5 days' duration; no discomfort at this time; L. M. P. was October 3, 1947; E. D. C. July 10, 1948.

Family History: Negative.

Physical Examination: revealed an extremely nervous, apprehensive, woman in severe pain. The pain was aggravated even by contact with the bed clothes. Mucous membranes were anemic and the sclerae were deeply icteric. Temperature was 100° F., blood pressure, 120 systolic, 90 diastolic, pulse 110, and respirations 26. The lips were dry and fissured. The heart and lungs were normal. There was some tenderness over the spleen and liver, but no enlargement of these organs. The kidneys were negative to palpation. The uterus was midway between the umbilicus and the ensiform. Fetal heart sounds were found in the lower left quadrant, rate 144 per minute. The fetal head was above the pelvic brim. There was no edema of the legs but a slight edema of both ankles. There were no leg ulcers or scars.

Laboratory Findings:

	Hgb.	*R. B. C.*	*W. B. C.*	*Poly.*	*Lymph.*
5–24–48	8 Gm.	3,000,000	14,950	76	23
5–27–48	8 Gm.	2,400,000	15,000	98% Sickling	55% Erythroblast
5–29–48	5 5 Gm.	1 990,000	17,500	72	22

5–25–48

Alkaline Phosphatase	7 units	Bilirubin	7.5 mg.
Icterus Index	40	Cholesterol Esters	55%
Serum Protein	7.47	Cholesterol	143
Serum Albumin	2.50	Hematocrit	28%
Serum Globulin	4.97	Blood Platelets	358,000

Fragility Test: Beginning 30
Complete 20
Markedly increased resistance to hypotonic salt solution.

The urine was practically negative. Her blood Wassermann and Kahn were negative and she had an Rh-positive factor. The sedimentation rate was 23 mm. in one hour.

X-ray of the heart and lungs was normal and x-ray of the skull showed a moderate amount of endostosis. The sella turcica was normal in size and configuration. X-ray of the pelvis and the long bones was normal. The electrocardiogram showed left ventricular hypertrophy.

From the above laboratory findings, a diagnosis of sickle cell anemia was made. The patient was given sedation and blood transfusions, 500 cc. each day for three days before the operation and 500 cc. after cesarean section; a total of 2,000 cc. of blood, also 100,000 units of penicillin daily.

On 5–29–48 the patient went in labor with very severe uterine tetanic contractions. The abdomen was of boardlike rigidity with extreme tenderness to touch. The fetal heart tones were rapid, 164 per minute. Rectal examination revealed an unengaged head. The cervix was long and thick and showed no evidence of dilatation. During the rectal examination, the

membranes ruptured spontaneously. The amniotic fluid was golden yellow in appearance.

It was felt that for the safety of the mother and baby, this patient should be delivered by cesarean section. Under local anesthesia, 1% novocaine infiltration was used, a cesarean section was performed and a living female baby was extracted, weighing 5½ pounds. The pain in the patient's abdomen and legs disappeared immediately on her return from the operating room. Her jaundice cleared completely by the sixth day and she made an uneventful recovery. The puerperium was afebrile and both the mother and baby were discharged from the hospital in the ninth postoperative day.

Discussion: This patient presents a typical case of an acute abdominal crisis in sickle cell anemia. The sudden onset of severe abdominal pain accompanied by nausea and vomiting with a tender abdomen and rigidity may simulate an acute abdominal condition. Stasis or sludging of the red cells may be present in the capillaries of various organs. The sickle cells encounter great difficulty in passing through the capillaries and the vascular changes are probably responsible for the many acute but temporary pains that occur. The pains in the joints with the elevated temperature may also be falsely diagnosed as acute rheumatic fever. Stiffness of the neck, headache, and vomiting may be mistaken for meningitis.

The diagnosis of sickle cell anemia depends on examining the blood of the patient for signs of sickling. The sickle cells can be demonstrated in a hanging drop preparation or by the sealed smear technique. The smears will also show reticulocytosis and normablastosis. Smears of the bone marrow will show a hyperplasia of the bone marrow with a reduction of fat cells. The extent of hyperplasia of the bone marrow, however, depends on the severity of the disease.

Prognosis: Individuals with the sicklemia trait may go through life without ever showing the active anemia. It does not prevent conception nor does it complicate pregnancy, labor or puerperium. When the active form of sickle cell anemia appears in pregnancy, then the prognosis is very poor for both mother and baby. Once the anemia develops, it does not completely disappear. There is always evidence of blood destruction. Pregnancy has a very unfavorable effect, in that it causes an exacerbation of the anemia.

Treatment: There is no specific treatment for this condition. Iron and liver are of no value. During the acute crisis blood and plasma should be given in adequate amounts, care being taken not to transfuse the blood from a donor showing the sickling trait. During remissions, the patient should be kept on a high protein diet with large doses of vitamins, especially A and B. This patient was given folic acid with small amounts of thyroid extract. The last time she was seen on 2–1–49 her hemoglobin was 11 Gms., R. B. C. 3,700,000 and she showed 40% sickling. Her basal metabolic rate was plus 10%. The baby showed 50% sickling.

Summary

1. A review of the current literature reveals only 27 cases of sickle cell anemia in pregnancy reported.
2. Another case of sickle cell anemia is reported.
3. In these 27 cases there was a total of 57 pregnancies.
   x: Sickle cell anemia in pregnancy is a very grave disease for both mother and baby. There was a maternal mortality of 26% and infant mortality of 52%.
4. Administration of penicillin and sulfa drugs to prevent and treat sepsis is important.
5. The treatment in addition to transfusions is a high protein diet, vitamins, folic acid, and thyroid extract.

REFERENCES

1. Herrick, J. G.: *Arch. Int. Med.* 6:517, 1910.
2. Killingsworth, W. P. and Wallace, S. A.: *South M. J.* 29:941, 1936.
3. Wintrobe, M. W.: *Clin. Hemet.*, 2nd Ed., Phila., Lea & Febiger, 1946.
4. Kracke, R. R. and Garver, H. E.: *Diseases of Blood and Atlas of Hematology* pp. 263–269, Phila., J. B. Lippencott Co., 1940.
5. Sydenst icke , L. P.: *J. A. M.* 83:12, 1924.
6. Yater, W. M. and Mallair, M.: *Ibid* 96:1671, 1931
7. Lash, A. F.: *Amer. J. Obst. and Gyn.* 27:791, 1934.
8. Richter, O. et al.: *Ibid.* 28:544, 1934.
9. Sharp, E. A. and Schleiter, E. W.: *Am. J. Clin. Path.* 6:580, 1936.
10. Lewis, A.: *Am. J. Obst. and Gyne.* 33:667, 1937.
11. Sodeman, W. A. and Burch G. E.: *New Orleans M. S. J.* 90:156, 1938.
12. Page, E. W. and Silton: *Am. J. Obst. and Gyne.* 37:38, 1939.
13. Kobak, Alfred J. et al.: *Am. J. Obst. and Gyne.* 41:811–823, 1949.
14. Spivak, M.: *Am. J. Obst. and Gyne.* 50:442–446, 1945.
15. Noyes, R. W.: *Am. J. Obst. and Gyne.* 52:469–473, 1946.
16. Martinek, Richard E.: *Am. J. Obst. and Gyne.* 53:332–334, 1947.
17. Carangelo, John and Otts, O. M.: *South. M. J.* 40:1016–1019, 1947.

THE MONTEFIORE HOSPITAL, 5TH AVENUE AT DARRAGH, PITTSBURGH 13, PA.

The Sickle Cell Disease of Negroes

Its significance for the bloods of other peoples

PAUL M. NEUDA, M.D.*

Sickle cell disease of the Negro (which designation includes both sickle cell anemia and the so-called sickle cell trait, the sickling condition without anemia) was not considered of ranking importance until recently although the number of affected Negroes in this country is of the magnitude of at least 1,000,000. The reasons for this attitude of the profession were mainly three:

1. The recognition of an existing prejudice against this disease, since it is found exclusively in the Negro and is regarded as a racial disease.

2. The fact that the greater incidence of this condition exists in the form of the so-called sickling trait, which is the sickling condition without a subsequent anemia. This "trait" however is considered merely an abnormality and not a disease: the blood of a Negro with the sickling trait is even regarded as fit for transfusion purposes.

3. The fact that nothing was known in sickle cell disease to warrant a general or even a broader interest of the profession in this disease of the Negro.

Much work has been devoted by hematologists and pathologists, to this strange blood condition. Primarily, the red cells of Negroes harboring the sickling tendency, do not differ in appearance from the erythrocytes of whites and healthy Negroes. If however, a droplet of such blood is placed on a slide and covered with a cover slip, then while anoxia develops, the original discoid form of the cells changes shape to a crescent-like figure, called a sickle. Depending on the acuteness of the sickling tendency, greater in sickle cell anemia than in "trait," this change takes place after hours or days. Many valuable details relating to the morphology and pathology of this change, are found in the literature. However, the etiology, the pathogenesis and the therapy of sickling, are not known.

In 1945 a new approach and finding were reported.[1] A substance, which is a normal physiological component of normal feces and has never played any pathological role in human disease, namely the Blood-Group-Enzyme (BGE), also called the "blood-group-property-destroying-factor,"[2] was shown by Neuda to accelerate the appearance of the sickled cells in sickling bloods, from hours and days by the usual standard method, to some few minutes. This fact proposed on the one hand, a rapid method for diagnosing sickle cell disease, but offered on the other hand, the first foothold on a substance acting possibly as a specific in the origination of the

*From the Laboratory for the Study of Human Blood. The Robinson Foundation, Inc., New York 5, N. Y.

sickling phenomenon. According to this latter view, either the BGE or a substance closely related to it, might be the aggressive agent contained in the plasma and fixing itself to sensitive red cells. This form of attack offers the enzymatic approach.

Although further verification of this view is awaited, the proposal of the hypothesis of the enzymatic basis of the blood destructive process underlying the sickling condition, has led to some highly practical further consequences. From the beginning, the generally accepted concept that sickle cell disease is a racial disease of the Negro, seemed rather an unfortunate idea, closing the door to a broader understanding and application of the peculiar pathology of this condition. The introduction of new terms, therefore, was needed to denote the changed situation in concept. The red cell reaction of the Negro to the BGE, leading to the accelerated sickle appearance was called "red cell sensitivity"; and the blood destruction itself was called "hemolysis of the sickle cell type," denoting by this term the apparent *type*-character of this disease, anticipating other anemic conditions of the same type.

This concept suggested two further tasks:

1. To find "red cell sensitivity" (which in Negroes was the "sickling phenomenon"),[3] in other people, and especially in Caucasians, or to rule it out.

2. To make the possible role of the BGE (or a substance closely related to it), and especially its origin in the blood, better understood in the general framework of the pathology of this disease.

The first question has been definitely answered by a paper recently published.[4] According to the findings therein reported, the idea of sickle cell anemia as a racial disease, can no longer be maintained. The same type of blood destructive process, the same red cell sensitivity to the BGE, though in another form, called "block-partition," exists in Caucasians. And block-partition is also found in non-sickling Negroes.

The second task requires further study of the sources of the BGE in the human body as to the possibility either of: a) its passing from the bowels into the blood stream, or b) its autochthonous formation within the blood stream.

After the finding of block-partition in Caucasians and non-sickling Negroes, as an apparent analogue of sickle formation in the sickling Negro, a third example of the same type blood destruction was found, confirming once more the type-character of this hemolysis. It is a new figure, also crescent shaped, occurring in both Caucasians and Negroes, with cirrhosis of the liver as the prototype disease. It is described in detail elsewhere.[5]

Important information as to the character of the blood destructive process operative in the sickling phenomenon, came recently from Pauling and his associates.[6] They found by means of electrophoretic studies that the hemoglobin in sickle cell anemia is fundamentally different from normal hemoglobin. They consider the cause of this abnormality to reside in a

molecular change in the globin part of the hemoglobin molecule and suggest that other hereditary hemolytic anemias should also be examined for the presence of abnormal hemoglobins.

Pauling's suggestion that the abnormal hemoglobin of sickle cell anemia in various conditions and in various people without regard to race may also be found by the use of the electrophoretic method, in other hereditary hemolytic anemias, would seem to be further indicated by Neuda's studies of BGE's hemolytic character.

The concept that the BGE or a substance closely related to it, might be the responsible pathological agent of all blood destructive processes of the sickle cell type, brings nearer to solution a number of highly interesting and practically important questions and raises others. Laboratory work on these questions is in continuous progress. To round out this picture of a new type of hemolysis, of a new disease and of a new pathologic agent, some of the following questions occur:

1. The assumption of the racial character of sickle cell disease, was from the beginning and would have remained, a serious obstacle to its understanding. Why should a part of mankind have a disease which another part in principle does not have? The fact is that the same type of hemolysis as found in the sickling condition of the Negro, exists in Caucasians. And this is only logical. Since the BGE is a normal component of the feces without regard to race, and also the blood group property of the red cells (A, B, O), upon which the BGE apparently acts, is common to all races, there would appear from the beginning to have been no reason for believing that this condition should not exist in all the people of the world.

2. The cause of the sickling phenomenon was never known. Known by observation only, was the fact that anoxia would cause the red cells of the Negro, endowed with the sickling quality, to assume a crescent form. This deadlock was broken by the finding of a specific substance, BGE, which greatly accelerates sickling, and by the evidence that BGE does not exist solely in the plasma of Negroes and does not produce solely the sickling phenomenon.[3, 4] The conclusion that the specific factor in the plasma combines with another specific factor in the red cells (presumably the blood-group substance) and the logical further assumption that the spontaneously sickling red cells contacted the aggressive substance, already present in the blood stream, would follow.

3. A most interesting aspect of sickle cell anemia has always been the peculiar cell fragility connected with the sickling process. Of greater significance than cell fragility, however, is the presence of an aggressive substance in human blood. If no such substance were present in the blood, red cells endowed with a predisposition to sickling, could not sickle. On the other hand, this substance might be present in the blood without being met by sensitive red cells.

4. And if such an aggressive substance were found in human blood, what should we do about it regarding blood transfusions? Here we

have a new and apparently pathologic agent in plasma, which may or may not combine with red cell sensitivity. When transfused, however, it might meet sensitive red cells. Are we to tranfuse blood of Negroes with the sickling "trait," because "trait" is generally today regarded more as an abnormality than as a disease? And should we not be aware of the danger in transfusions by a substance which in fact is blood-group-property-destroying[2] and might well attack, by its very property, not only red cells but even tissue cells? What about toxic hepatitis, for instance? These and similar questions demand answers.

Sickle cell disease of the Negro was the starting point of this investigation on a new enzymatic type of hemolysis. It will remain a most important object of study on this condition. The results, however, will be of benefit to our knowledge of blood pathology in various conditions and in all the races.

Summary

1. Sickle cell disease appears to be the first, though the best example of a new, probably enzymatic type of red blood cell destruction, which however exists also in conditions other than sickle cell disease, and is found in people other than Negroes.

2. Block-partition is another example of the kind of blood destruction found in sickling Negroes. It occurs in Caucasians and in non-sickling Negroes as well. It is an apparent analogue of the sickling condition and not confined to race.

3. There is evidence of still another form of this same type of blood destruction.

4. The common red cell reaction on the slide, to the blood-group-enzyme in various hemolytic processes, suggests their inner relationship, and further suggests that this enzyme, or a substance of a similar nature, plays a prominent role in the pathology of these various conditions.

5. The possibility of the presence of the blood-group-enzyme (or a similar substance) in human blood, with or without a concomitant major red blood cell destruction, is of paramount interest.

6. The technique of blood transfusion should take into account the possible presence in bloods, of a substance, which, as its name (blood-group-property-destroying-factor) reveals, attacks the blood-group property.

7. A thorough study of this enzymatic substance and of the causes of its appearance in blood, is indicated.

Bibliography

1. Neuda, P. M. and Rosen, M. S.: Preliminary report on a rapid method for diagnosing sickle cell disease. *J. Lab. and Clin. Med.* 436–438, 1945.
2. Schiff, F. and Boyd, M.: Bloodgrouping technic. *New York: Interscience*, 1942.
3. Neuda, P. M.: Red blood cell sensitivity to the blood-group-enzyme. *Science* 296–297, 1947
4. Neuda, P. M.: Red blood cell sensitivity in Caucasians. *Ann. of Int. Med.* 1024–1029, 1949.
5. Neuda, P. M.: A new pathological red cell figure in human blood. (In Process)
6. Pauling, Linus Itano, A., Harvey, Singer, S. J. and Wells, Ibert C.: Sickle cell anemia, a molecular disease. *Science* 543–548, 1949.

Fishbone Perforation of the Intestines

A review of the literature and report of a case

MARIO A. CASTALLO, A.B., M.D., Sc.D., F.A.C.S.*
AND
BASIL G. GILETTO, M.D.**

In reviewing the literature on fishbone perforation of the gastro-intestinal tract, we have been able to collect 54 cases. Undoubtedly there have been innumerable other cases in which the cause of perforation was not discovered due to the fact that the bone was inadvertently removed with pus or other debris at operation, or to the further migration of the bone. The fate of an ingested fishbone in the body depends upon several factors including the size, shape and consistency of the bone, as well as the contents of the digestive tract, and peristaltic activity of the bowel when the bone is ingested.

Knud Faber[10] contributed a study on ingested fishbones, of interest because it was undertaken on a group of Scandinavians, living in a country where fish constitutes one of the chief articles of diet, and because many of the subjects were inmates of an institution for the feebleminded, who are more likely not only to ingest such bones accidentally, but who also frequently swallow sharp objects intentionally. He found that in a large number of cases, innumerable fishbones pass through the gastro-intestinal tract causing no harm whatsoever. Occasionally, if swallowed in large numbers, the bones may form a mass in the cecum or rectum, giving rise to intestinal disturbances including obstruction, diarrhea and hemorrhage. The reason for the relatively innocuous course of these sharp objects is that the bones may be partially digested by the hydrochloric acid of the gastric juice. It is well known that in carniverous animals who swallow their victims whole, such as the boa constrictor, after the time required for digestion has elapsed no trace of any tissue of the devoured animal is to be found in the digestive tract except hair. All bone has disappeared. Experimentally, Faber[10] demonstrated that fishbones immersed in hydrochloric acid of a concentration similar to that found normally in the gastric juice, soon became soft and transparent. He concluded that cases of perforation in which the bone has evidently retained its consistency even after having passed through the stomach must be due to diminished acidity or anacidity of that organ. In this event, the bone persists hard and pointed and acts as any other sharp pointed body. If the bone is thin and of about uniform

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thickness throughout its length, it will then probably cause an acute perforation.

Exner[9] has shown that needles in the intestine show a tendency to turn the blunt end downward. Whether this applies also to pointed bones and other pointed objects is not known. Once in the intestine, the bone may be covered by or buried in the intestinal contents, which thus shield the intestinal wall from injury. If the intestinal content is fluid, and peristalsis violent, the sharp point of the bone may be forced through the wall of the intestine. If the bone is more than 12 cm. long, it will be too long to execute the normal turns of the intestine without becoming lodged, or perforating the wall of the intestine. Even smaller bones may have a similar fate in the presence of adhesions or pathologic strictures or diverticula of the intestine.

Occasionally, a bone is covered with the food bolus and is passed onward with it to be expelled through the natural channels. It may, in passing, injure the mucosa with resulting inflammation or ulceration. If the bone is very fine and thin, it may perforate the intestine through a very tiny orifice, which closes up after it, and pass on into the abdominal cavity or migrate into or through other organs. With the aid of the movements of the abdominal muscles in constant action during digestion and respiration, the bone may finally work its way to the surface of the skin, usually about the umbilicus, and leave the body by this route, as a rule through a superficial abscess. In such cases there may be no trace of its passage through the intestinal wall. According to Jackson and Jackson[18], however, "fishbones do not generally penetrate by direct puncture but ulcerate through by prolonged sojourn after lodgement in one location." They emphasize the fact that this takes time, which may have a lifesaving significance.

The most common sites for lodgement of foreign bodies in the small intestines are the terminal ileum, Meckel's or other diverticula, the jejunum and rarely in the appendix. According to Ginzburg and Beller,[14] who have reported a series of twelve cases of perforation of the large intestines by non-metallic foreign bodies, including six fishbones, arrest of such bodies in the small intestines is rare. They describe the first effect of the foreign body on the mucosa as an abrasion with ulcer formation. Necrosis then progressively involves the layers of the intestinal wall, and they believe that infectious organisms from the intestine may pass through such areas even before definite perforation has taken place, causing local peritonitis. It is suggested that obscure cases of pericolitis and omentitis might be due to foreign bodies, which having produced such a lesion, dropped back into the lumen of the intestine to be eliminated with the feces. Attention is also drawn to the possibility of purulent thrombophlebitis developing, following perforation of the bowel along the mesenteric border. The authors also reported a case by Benda in which the fishbone was discovered in the superior mesenteric vein with thrombosis of the ileo-colic branch. According to Exner's 9 experiments, the intestinal mucosa reacts to contact

with foreign bodies by a special contracting mechanism forming deep folds and a transitory anemia lasting for five to fifteen minutes.

Complications that may be expected if the bone does not pass out of the bowel in the manner described, include enteritis, ileus, hemorrhage, chronic perforation with abscess formation, or acute perforation with diffuse peritonitis.

Wölfer and Lieblein[44] collected 1,184 cases of foreign bodies in the gastrointestinal tract. Of 58 cases in which the foreign body consisted of bones, 5 died. The foreign body was discovered incidentally at autopsy in 3 cases. Abscess formation was noted in 11 cases and the foreign body was eliminated through the natural channels in 34 cases. In two cases, death was due to perforation of the intestine with resulting peritonitis, in one case to gastric hemorrhage, and in two cases to intestinal hemorrhage. They too, mention the tendency of ingested fishbones to conglomerate into tumor-like masses. Diagnosis of ingested fishbone is of course most difficult unless the patient remembers having eaten fish. Jackson[18] emphasizes the importance of daily observation and palpation. Stools should be washed and passed through a sieve. In cases of sharp pointed objects, he recommends daily roentgenographic localization by anteroposterior and lateral roentgenography as a basis for counting days of lodgement. A long fishbone will jam in the turns of the duodenum. Unless lodged or perforating, a foreign body is likely to be passed within 72 hours after ingestion. His rule is to wait four days and if the foreign body has not become dislodged, he operates. Tenderness is an indication for immediate operation.

According to Wölfler and Lieblein,[44] ten per cent of all patients with foreign bodies remaining in the gastrointestinal tract die. In the present series of 54 cases, 14 patients died, placing the mortality rate for perforation of the small intestines at 25.9%. This series included 17 cases of perforation of Meckel's diverticulum by fishbones, which according to Persson[36] has a much better prognosis than perforation of this area due to any other cause such as diverticulitis. In the 17 cases here included there were four deaths.

Ginzburg and Beller[13] have classified cases encountered surgically into the following groups: (1) acute peritonitis; (2) localized intra-abdominal abscess; (3) intra-abdominal inflammatory tumors; (4) inflammatory tumors of the abdominal wall; 5. abscess of the abdominal wall; and (6) inflammation and obstruction in a hernial sac.

In patients with peritoneal symptoms, operation must not be delayed. There were nine such cases in the present series, with four deaths. In cases with localized intra-abdominal abscess (seven such cases in present series) differential diagnosis from other intra-abdominal conditions must be made. Ginzburg and Beller[13] emphasize the fact that in cases of this type, when the bone is found, no effort should be made to locate the site of perforation, because the latter will as a rule, have closed spontaneously. With tube or rubber dam drainage and adequate drainage of the superficial layers of the wound, healing may be expected within a few weeks. With the use of the

sulfa drugs, penicillin, and blood transfusions, cures may be expected in a greater majority of cases.

Careful differential diagnosis is required to avoid errors in treatment and prognosis. Such a case was described by Wagner[25] in which the differential diagnosis included consideration of an ovarian tumor with twisted pedicle, old paratyphlitic exudate, and sarcoma of the abdominal wall. It has been emphasized that inflammatory tumors of this type are usually smooth and firm and frequently the mass of the tumor is formed by inflamed omentum.

Treatment will, of course, depend upon the condition produced by the bone. In cases of acute perforation, immediate operation is indicated to prevent death from diffuse peritonitis. In cases of chronic perforation with abscess formation the type of operation will depend upon the location of such an abscess. According to Persson[26], when Meckel's diverticulum is the site of perforation it should be resected. Gangrenous segments of the bowel would also require resection.

In all reports males seem more likely to suffer from intestinal perforation due to foreign bodies than do females, possibly owing to less careful eating habits. In the present series there were 37 males and 15 females, 2 unclassified. One patient was 8 years of age. Of the remainder, three were from 10 to 20 years, 8 from 20 to 30 years and 17 from 40 to 50 years, 7 from 50 to 60 years and 3 from 62 to 66 years of age; 15 were unclassified as to age. In 3 cases the preoperative diagnosis was appendicitis. Other conditions suspected included peritonitis, ovarian cyst, gallbladder disease, lymphadenitis, pancreatitis, paratyphlitis, sarcoma of the abdominal wall and intestinal obstruction, carcinoma of the stomach with metastasis to the liver, pericholecystic abscess and perforating neoplasm.

Report of a Case

Patient: White, C. Z. Aged 63, female, was admitted to St. Mary's Hospital with a diagnosis of ruptured appendix with generalized peritonitis. The patient was in the hospital for a total stay of nineteen days. The chief complaint on admission was pain in the lower abdomen. The patient said that about 4:00 P. M. she developed a sudden sharp pain in the lower right quadrant. The pain became more severe and nausea with vomiting started with gradual generalized pain throughout the whole abdomen. The family physician was called about 11:00 p. m. and after examination he said she had all the physical findings of a ruptured appendix with generalized peritonitis. The patient complained to the physician that she felt something sticking her like a needle, and localized the pain around the area of McBurney's point. Not much significance was attached to this complaint. The family physician immediately recommended that she be admitted to the hospital for operation. After operation, when questioned, the patient definitely remembered that on the previous day she had ingested a fishbone. It had lodged in her throat but she was able to swallow it down and thought nothing more of it. The pain developed

almost twenty-three hours after swallowing the fishbone. This was only ascertained after the patient had been told that a fishbone had been removed from her intestine.

Past medical history is of no significance except that she had thirteen full-term pregnancies with eleven living children. There was no unusual illness or operation throughout her life. Her family history was irrelevant.

The physical findings at the time of admission were as follows: temperature 100.2, pulse 120, respirations 24, BP 145/80. A catheterized urine specimen was negative. CBC was as follows: Hb. 80%, RBC 4,430,000, WBC 13,250; polys, 79%, lymphs, 19%, monocytes 2%. Physical examination revealed a normal well-developed woman, who complained of excruciating abdominal pain.

The following positive signs were present. The abdomen was tense, there was pain on palpation and tenderness throughout the whole abdominal area. There was definite rebound phenomena and referred pain. There was marked excruciating pain over McBurney's point where the patient claimed that she felt a needle pinching her from the inside.

The patient was prepared for operation and operated on one and a half hours after admission. She received gas and oxygen, straight ether anesthesia and oxygen. After the proper preparation a right rectus incision was made. On opening the peritoneal cavity the entire peritoneum was red and a generalized spreading peritonitis was present. Exploring for the appendix the small bowel was encountered and found to be large and inflamed with a foreign body protruding. The foreign body was held with a forceps and the bowel opened with the cautery and the foreign body removed. The bowel was then closed with No. 0 chromic catgut; two rows of sutures were employed in the area. On opening the tissue containing the foreign body, it was found to be a fishbone about three centimeters long. (Fig. 1)

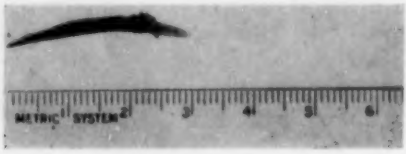

FIGURE 1

The fishbone was found to have entered the bowel at a ninety degree angle, definitely perforating through and through. (Fig. 2) The appendix was located, localized and found to be inflamed in its outer tip. This was removed.

The patient returned to her room in fair condition. She received nothing by mouth for 48 hours. Wangensteen suction was employed as soon

FIGURE 2

as the patient returned to her room. She was given 2500 cc. of fluids, 1500 cc. of 5% glucose and 1000 cc. of 5% glucose in distilled water. The patient was also given 75 mg. of sodium sulfadiazine intravenously daily. Wangensteen suction was employed for three days at which time the patient was able to retain fluids. The first 24 hours the temperature, pulse and respirations were as follows: temperature averaged about 102, pulse 112 and respirations 28. Seventy-two hours postoperatively the temperature was 99, pulse 88 and respirations 24. From then on the temperature averaged about 99 to 100, then gradually became normal and the patient had an uneventful convalescence from her symptoms. She was out of bed fourteen days postoperatively and left the hospital on her nineteenth day in good condition and was referred back to her family physician.

The patient was seen six weeks after operation. The incision had completely healed and she was in good health.

CONCLUSIONS

1. A case of fishbone perforation of the small intestine is reported with 54 other cases reported in the literature.
2. Preoperative diagnosis is often difficult and is usually made after laparotomy.
3. The symptomatology is very variable.

4. **Many cases must occur other than those reported in the literature. In most instances the fishbone must either be digested or pass through the alimentary tract without symptoms.**

We wish to thank Dr. Frank McCauley for permission to use the data on the case reported and Dr. John M. Stack for his perusal of some of the literature for the authors.

BIBLIOGRAPHY

1. Armitage, G.: Three perforations of the ileum caused by fishbones. *Brit. M. J.*, 1:307, 1928.
2. Bacon, T. and Corliss, P. G.: Perforation of the intestine by a fishbone. *New England J. Med.* 204:214–15, Jan. 29, 1934.
3. Bearse, C.: Fishbones as a cause of intestinal perforation. *New England J. Med.* 201:885–6, Oct. 31, 1929
4. Blanc, H. H.: Contribution à la pathologie du diverticule de Meckel. *These. de Paris*, 1899.
5. Bunch, G. H., Burnside, A. F. and Brannon, L. J. (5 cases): Perforation from ingested fishbone. *American J. Surg.* 55:169–172, Jan. 1942.
6. Carp, Louis: Foreign bodies in the intestines. *American J. of Surg.* 2:169, 1927.
7. David, I.: An errant fishbone. *British Med. J.* 1:106, 1918.
8. Doughlas, J.: Perforation of the duodenum and liver by a fishbone. *J. A. M. A.* 95:265–6, July 26, 1930.
9. Exner, A.: Wie schützt sich der Verdauungstract vor Verletzungen durch spitze Fremdkörper. *Arch. f. d. ges. Physiologie des Menschen und der Thiere*, 89:233, 1902.
10. Faber, K.: Om Fiskeben som Fremmedlegemer i tarmen. *Hospitalstid.* 4 R. VI:429, 1898.
11. Fry, W.: Fishbone perforating the caecum. *Virginia M. Monthly* 66:173, 1939.
12. Fürstenau, J.: Ueber Darmperforationen durch verschluckte Fischgräten. *Deutsche med. Wchnschr.* 55:1135, July 5, 1929.
13. Ginsburg, L. and Beller, A. J.: Non-metallic perforation of intestines by foreign bodies. 6 Cases. *Ann. Surgery* 86:928, 1927.
14. Golberg, S. L. and Levan, A.: Fishbone perforation of the jejunum. *American J. Surgery* 58:446–447, Dec. 1942.
15. Hagler, F. and Stewart, J. W.: Acute perforation of Meckel's diverticulum by foreign body (fishbone). *J. A. M. A.* 14:259, 1920.
16. Henrichsen, A.: Ueber einen Fall von Fremdkörperdiverticulitis. *Münch. med. Wchnschr.* 68: 1423, 1921.
17. Hertz, J.: Perforation de l'appendice par une arête de poisson. *Bull. et mem. Soc. anat. de Paris* 87:239, 1912.
18. Jackson, C. and Jackson, C. L.: Foreign bodies in the intestines. *Med. Record* 140:285, Sept. 19, 1934.
19. Jones, J. F. X.: Incarcerated hernia in man aged 93 with fishbone in appendix. *New York M. J.* 116:670, 1922.
20. Karpinski: *Gaz. lek.* No. 41, 1841
21. Lichtenstein, M. E.: Meckel's diverticulum. Peritonitis due to perforation by fishbone. *Quart. Bul. Northwestern Univ. Med. School* 15:296, 1941
22. Lötzow-Holm, G.: Tre tilfaelde av tarmperfortion paa grund av fiskben. *Norsk. Mag. f. Laege vidensk.* 83:523–6, 1922.
23. Melville, C. B.: Perforation of small intestines by swallowing foreign bodies. *Australasian & New Zealand J. Surg.* 10:146, 1940–1.
24. Morton, C. A.: Large intra-abdominal abscess from a foreign body which had perforated the bowel. Drainage and recovery. *Brit. M. J.* 1:241, 1894.
25. Norinder, E.: Perforation by fishbone with formation of liver abscess. *Acta. chir. Scandinav.* 76:136–46, 1935.
26. Persson, T.: Meckel'sches Divertikel durch eine Fremdkörper perforiert. *Acta. chir. Scandinav.* 82: 330, 1939.
27. Piquand, G. and H. Grenet: Perforation d'un diverticule ileal par un arête de poisson. *Bull. et mem. Soc. anat. de Paris* 2:390, 1900.
28. Pye-Smith, R. J.: A case of acute intestinal obstruction caused by a fish fin, lodging above a Meckel's diverticulum. Laparotomy; rapid recovery. *Lancet* 1:472–4, 1889.
29. Rossmann, J. L.: Fishbone perforation of a Meckel's diverticulum. *Canad. M. A. J.* 49:48–9, 1943.
30. Stetten, DeWitt.: Extrusion of ingested fishbone at umbilicus. *Ann. Surg.* 85:306, 1927.
31. Stone, H. B. and Owings, J. C.: Perforation of stomach by a fishbone. *American J. Surgery* 25: 180, 1934.

32. Tamraz, J. M.: A case of acute gangrenous diverticulitis (Meckel's) with perforation due to fishbone. *Mil. Surgeon* 87:328, 1940.
33. Taylor, G. and Lakin, C. E.: A fatal case of phlegmonous inflammation of the duodenum following impaction of a fishbone. *Lancet*, 2:224–5, July 22, 1911.
34. Tlach, E.: Beitrag zur Kasuistik der durch Fremdkörper des Magen-Darmkanals, insbesondere Fischgräten, Verursachten Bauchwandabscesse und Bauchwandfisteln. *Diss. Breslau*, 1920.
35. Wagner, A.: Entzündlicher Bauchdeckentumor, hervorgerufen durch einen aus dem Darm durchgebrochenen Fremkörper. *Munch. med. Wchnschr.* 49:1919, Nov. 18, 1902.
36. Walkling, A. A.: Perforation of Meckel's diverticulum by a fishbone. *Ann. Surgery* 93:298, 1931.
37. Webb, R. C.: Meckel's diverticulum perforated by a fishbone. *Ann. Surgery* 98:159, 1933.
38. Weinstein, V. A.: Fishbone perforation of Meckel's diverticulum. *J. Mt. Sinai Hospital* 9:29–32, 1942–3.
39. Wettergren, C.: Abscess of the liver containing fishbone swallowed 4 years before. *Svenska Läk.-Sällsk. Sammank.* 1880, p. 37; also M. Herald, *Louisville* 3:10, 1881–82.
40. Wilcox, A. E.: Perforation of Meckel's diverticulum by a fishbone. *Minnesota Med.* 15:558, 1932.
41. Wilhelm, E.: Hepatic abscess caused by fishbone. *Zentrab. Für Chirurgie*, 52-1464, July 1925.
42. Williams, R. V.: Perforation of Meckel's diverticulum by fishbone. *Minnesota Med.* 23:44–66, 1940.
43. Winge, E.: Pyelephlebitis fremkaldt ved fiskeben. *Nord. med. Ark.* 12, no. 27, p. 5. 1880.
44. Wölfler, A. and Lieblein, V.: Die Fremdkörper des Magen-Darmkanals des Menschen. Stuttgart, Enke, 1900. (*Deutsche Chir. Lfg. 46 B.* 402 pp.)

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"You have done more for me in an hour," said the veteran, "than all the psychoanalysts could do for me in a life time."

Figure 1 illustrates a most striking case of what can be done to the face through a simple vermilion border incision. A good rule to insure lateral equality in cutting the skin is to first make the incision at a certain distance from a landmark. This is done by spearing the skin with a bayonet blade at critical points and then connecting them into one incision.

FIGURE 1

Figure 2 is a diagram of this feature. Figure 3 shows method of stripping skin with the blade "up" as taught to his pupils by the illustrious Charles H. Mayo.

Figure 4 shows two middle stitches of 4–0 chromic catgut about a third of a centimeter deep on atraumatic needle, for the purpose of double-thickening the upper lip, and increased exposure of lower lip.

Figure 5 shows the completion of 5–0 plain atraumatic interrupted suturing.

This patient became a bridge tender because of complexes about facing the public. When he came to the office a week later when the "after" snapshot was taken (Polaroid equipment), he said, "At a lunch counter a girl actually spoke to me." He now has the fortitude to seek employment commensurate with his ability.

In addition to thickening the lip and increasing the visible vermilion surface this operation tends to improve appearances by lifting the corners of the mouth and lessening heavy smile lines.

139 SOUTHEAST THIRD STREET, MIAMI, FLORIDA

Treatment of Obesity

S. C. Schmidt, M.D.

Obesity, one of the most common metabolic irregularities, estimated to be present in 1 out of every 5 adults in the United States,[1] is defined as an exaggeration of the normal process of fat storage. Persons who are 20% above normal, or average, weight, based on sex, age, height and bone structure (small, medium or large), are considered by American life insurance companies as being overweight. McLester[2] states than an excess of 25% over the individual normal definitely constitutes obesity.

Pathologic Conditions Associated with Obesity

The statistics of life insurance companies prove that obesity tends to shorten the life span, and many investigators have observed pathologic conditions associated with excessive weight. Danowski and Winkler[3] observed an unusually high incidence of varicose veins, diseases of the joints (mostly of the knees and ankles) and hypertensive cardiovascular disease in obese persons. According to Preble[4] the mortality rate from cardiovascular diseases is two and one-half times greater in overweight persons as in those of normal weight. There have been noted in obese patients a tendency toward the development of diabetes mellitus and a predisposition to gallbladder disease, gout and chronic bronchitis. Obesity constitutes an additional risk in surgery.

Classification and Causation

In accordance with Beardwood's[5] differentiation of types, obesity may be classified as follows:

1. Exogenous (due to hyperalimentation)
2. Endogenous
   a. Endocrinopathic origin (dysfunction of the pituitary, adrenals or gonads)
   b. Nervous origin (due to injury or disease of the diencephalon)
   c. Hydrolipomatous (defect in salt-water metabolism, but not "true obesity")

Conceptions of various authors with respect to the causes of obesity vary widely. While some[6,7] attribute all cases of obesity to overeating, others stress psychosomatic[8,9,10] or heredofamilial[11] factors. It has been stated[12] that, while overweight is impossible without excessive caloric intake, overeating is often not the primary cause of the condition but a result of other factors.

Dietary Treatment

Whatever the contributing factor, obesity could not be present without excessive ingestion of food, either absolute or relative. It follows, therefore, that successful treatment of obesity can be achieved only with dietary restriction.

Effective management of obesity depends on the solution of several therapeutic problems:

1. Appropriate reduction diets
   a. Low caloric
   b. High protein
   c. Low carbohydrate
   d. Low fat
   e. Low salt
   f. Large bulk diets, supplemented with vitamins or iron-liver preparations if required
2. Maintenance of the patient's will to adhere to the diet
   a. Psychotherapy (encouragement)
   b. Medication (anorexigenic preparations)
3. Correction of endocrinopathic disorders (mostly hypothyroidism and hypogonadism)

Reducing diets, with respect to caloric values, should be adjusted to individual requirements. Patients with sedentary occupations require fewer calories than those who do hard manual labor. For either group, the caloric intake should be well below the output. Most reducing diets permit between 600 and 1,500 calories a day. Wohl[13] allows an intake of about 20 calories daily for each kilogram of ideal body weight, while McLester[2] limits the daily ration to 30% to 50% below the calculated maintenance figure.

The therapeutic objective of reducing diets consists in eliminating by oxidation excessive body fat while protecting the body proteins. Maintenance of the nitrogen equilibrium is therefore of paramount importance. Proteins not only possess a high specific dynamic action but are, according to Bauer[14] more easily oxidized than carbohydrates. It follows that reducing diets must be high protein diets. McLester[2] recommends 1.5 to 1.7 Gm. of proteins per kilogram of calculated ideal body weight. Lean meat, fish, eggs, milk and cheese provide excellent sources of proteins of high biologic value.

In order to preserve the nitrogen balance, at least 50% of the energy provided by the diet should be supplied by carbohydrates. A level of about 0.75 to 1.5 Gm. of carbohydrates for each gram of protein, preferably in the form of fruits and vegetables, is necessary to insure an acid-base equilibrium, to avoid the dehydration of acidosis or the hydrophilic tendencies of the tissues in alkalosis. According to Bauer,[14] the diet should contain

not more than 85 Gm. of carbohydrates daily. The fat level in reducing diets should be very low; 20 to 30 Gm. of fat meet all biologic requirements and insure full utilization of fat-soluble vitamins.

Many cases of obesity are complicated by defective salt-water metabolism, with consequent retention of water. Restriction of sodium chloride intake is therefore suggested by many writers. Bauer[14] recommends reduction of salt intake to 2 to 3 Gm. a day, which counteracts the tendency in obese persons toward storage of abnormal amounts of water in cutaneous and subcutaneous connective tissue.

To alleviate the feeling of hunger so common with restrictive diets, as well as to minimize constipation, reducing diets should contain a maximum of bulk, provided by green vegetables and fruits.[5] Their content of mineral salts and vitamins may be supplemented with polyvalent vitamins and, in cases of secondary anemia, with iron-liver preparations.

The best selected diet, however, will fail to produce results as long as the patient is unwilling, or lacks the determination, to adhere to it. Many patients are reluctant to diet because of their fear of ensuing weakness or other detrimental effects. Others do not have the will power necessary to override the gnawing sensation of hunger which so often accompanies restricted diets or to overcome the temptation offered by the sight or smell of food. Explanation and education by the physician are helpful, and encouragement and proper psychotherapeutic approach often bear fruit. However, in the majority of obese patients, the response to psychotherapeutic efforts alone is poor—the spirit only is willing, while the flesh remains weak.

Adjuvant Therapy

Anorexigenic Drugs—These patients, in the opinion of many well qualified authorities, should be granted the benefit of a "crutch," in the form of anorexigenic drugs, in conjunction with a restricted diet. Chemically, most accepted appetite depressants are aminopropanes. Their pharmacologic action causes: a) a depressing effect on gastric motility due to hunger (relaxation of the stomach and increase in the tone of the pylorus)[16], b) a stimulating effect on the central nervous system and c) a temporary rise in the metabolic rate.[17] Most aminopropanes begin to produce these effects in twenty minutes to one hour after oral administration, lasting six to twelve hours. In a few cases the duration has been up to thirty-six hours. In therapeutic doses, aminopropanes may affect the blood pressure, pulse rate and respiratory rate; however, the changes, if they occur at all, are only slight and temporary.

The delay in emptying time of the stomach causes a sensation of fullness after ingestion of moderate amounts of food; it reduces the feeling of hunger and eliminates the craving for food, thus enabling the patient to adhere to a restricted diet without feelings of self-sacrifice and self-pity. Gradually the stomach adjusts itself to the limited charge; it shrinks, and the patient may remain free from hunger sensation on a limited diet even after anorexigenic medication has been discontinued.

The stimulating effect of anorexigenic compounds on the nervous system itself in elevation of mood, a sense of increased efficiency and intensification of the urge to work. Fatigue is decreased and sleepiness is counteracted.

These reactions are of real benefit to the obese patient inasmuch as they combat the let-down feeling frequently experienced in reducing regimens. Furthermore, they help maintain full working capacity and fortify the patient's determination to adhere to the restrictive diet.

Side Effects—The most commonly encountered side effects from treatment with aminopropanes are dryness of the mouth and constipation, the latter as the result of the relaxing effect of the aminopropanes on the bowel. Insomnia has been observed in some cases when the drug was given too late in the afternoon. There have been some reports of mild tachycardia, hypertension and elevation of the metabolic rate after administration of single large doses. However, this tendency decreased appreciably when the drug was given continuously,[18] and some observers have stressed the fact that a decrease in blood pressure followed the reduction in weight.[19] Some patients exhibited a sense of exhilaration for a few days, and some hypersensitive patients reported a sensation of apprehension, depression or exhaustion. There have also been some complaints of headache, dizziness, halitosis, burning in the throat and heartburn. Nausea and vomiting have been reported as toxic reactions in hypersensitive patients or after excessive doses of anorexigenic compounds. These reactions could be controlled efficiently by decrease in dosage.

Contraindications—Contraindications for treatment with aminopropanes are relatively few. The drugs are definitely contraindicated in persons with hypersensitivity to ephedrine-like drugs; they should not be given to patients suffering from insomnia, pronounced excitability and excessive restlessness. Marked coronary impairment and conditions in which vasoconstrictors may not be used are also contraindicants. In cases of advanced hypertension or cardiovascular disease, great caution in treatment with aminopropanes should be exercised. Few cases of habituation to anorexigenic drugs have been reported[20]; nevertheless, the possibility, however remote, should be borne in mind.

Thyroid Therapy—A considerable number of obese patients show signs of hypothyroidism. However, a low basal metabolic rate alone does not always indicate a thyroid deficiency; metabolic rates as low as 20% have been reported in normal persons. Cautious thyroid medication, starting with small doses, will bring the basal metabolic rate to a normal level. It has been clinically demonstrated that small amounts of thyroid can be given many obese patients with normal metabolism, with good reducing effect and without harmful side action. The diuretic action of thyroid has a definite bearing on water retention in obese patients. Close medical supervision in all cases of thyroid medication is absolutely essential, and the patient should be kept on a restricted diet.

Compounds Investigated

As mentioned previously, most anorexigenic drugs are aminopropanes. The first compound extensively investigated was amphetamine sulfate. It has a long record of efficiency and low toxicity. The average dosage is 5 to 15 mg. given three times a day, one-half to one hour before meals. The dosage should be individually adjusted, and the preparation should not be given later than 4 p. m., because of possible interference with sleep.

An improvement in efficiency over the original amphetamine was found in its dextro-rotary isomer, *d*-amphetamine sulfate. It is administered in doses of 5 to 7.5 mg., three times a day, one-half to one hour before meals; its increased efficiency allows a decrease in dosage to about one-half of amphetamine sulfate.

Recently there have been some newer developments in aminopropane derivatives. Their anorexigenic effect is somewhat weaker; their interference with sleep, however, is less pronounced. The most important of them is *p*-methyl-amphetamine (*dl*-1 *p*-methyl-phenyl) 2-aminopropane. Its main advantage over other aminopropanes is that it does not interfere with sleep; it can therefore be taken as late as 9 p. m. Its anorexigenic effect, however, is far inferior to that of the other amphetamines. It has been administered safely in large doses; one author suggests administration of 10 to 15 mg. of *d*-amphetamine at 10 a. m., and 40 mg. of *p*-methyl-amphetamine at 4 p. m. and at 8 p. m.

Another anorexigenic drug closely related to the amphetamines is methamphetamine (desoxyephedrine) hydrochloride. The pharmacologic action and uses are the same as those of the amphetamines. Its central stimulating effect is somewhat greater, while the circulatory action is slightly less than that of the amphetamines. The average dose is 3 to 6 mg. daily; it should not be given less than four hours before bedtime.

In those cases in which endocrine disorders are the basic cause of obesity, correction of the glandular dysfunction should be attempted. In hypogonadism, treatment with gonadotropins or gonadal hormones may produce results.

Lakobie*—A New Reducing Compound

The trend in recent years has been in favor of combining the anorexigenic action of amphetamine drugs or amphetamine-like substances with the calorigenic and diuretic action of thyroid. Most of these reducing compounds contain varying amounts of phenobarbital to counteract such undesirable side effects as increased nervousness, hyperirritability and insomnia. These appetite-depressing compounds are designed as adjuncts to restrictive diets.

One of the newer reducing compounds is Lakobie. It has been under

*Pharmaceutical Distributors, Inc., Chicago, Ill.

clinical investigation for some time, and its dosage, mode of administration, efficacy, effect on blood pressure and pulse rate and its side effects and toxicity have been extensively studied in 75 unselected cases. Lakobie contains amphetamine hydrochloride, thyroid and phenobarbital, in three different combinations:

Lakobie 15—	Amphetamine hydrochloride	15 mg.
	Thyroid	30 mg.
	Phenobarbital	15 mg.
Lakobie 10—	Amphetamine hydrochloride	10 mg.
	Thyroid	30 mg.
	Phenobarbital	15 mg.
Lakobie 5—	Amphetamine hydrochloride	5 mg.
	Thyroid	30 mg.
	Phenobarbital	7.5 mg.

These combinations are the result of two years of investigation. During the pharmacologic and clinical studies it became obvious that several objections to the reducing formulas could be raised with respect to the amount of amphetamine hydrochloride in the more potent compounds (Lakobie 15 and Lakobie 10) as well as to the content of thyroid (30 mg.), in all three preparations.

In a discussion of the aforementioned objections, it should be stated emphatically that all patients treated with potent drugs like amphetamine and thyroid should be under the close and constant supervision of a physician. The modus of administration and the dosage can thus be regulated to suit the individual needs, and any harmful side effects can be avoided.

Amphetamine Content: Toxic reactions to amphetamine are extremely rare; in most of the cases in which such reactions have occurred, over-dosage has been the cause. Long clinical experiences documented by numerous authors have clearly demonstrated that amphetamines, even in large doses, can be given safely and for prolonged periods. Among writers who have reported prolonged use of amphetamine (amphetamine sulfate) in large doses are Harper (30 to 40 mg. daily),[21] Myerson (30 to 50 mg. daily),[22] Mathews (90 mg. daily)[23] and Hahne (100 to 240 mg. daily).[24] Bakst[25] gave 15 to 30 mg. daily for nine years; Ulrich[26] gave 20 to 40 mg. daily for two years; Myerson[27] administered 60 mg. daily for fourteen months; Bloomberg[28] prescribed 70 mg. daily for more than two and one-half years; Gorell[29] gave 80 to 110 mg. daily for two years; Korns[30] reported daily doses of 80 to 150 mg. for one year and again 150 mg. for six months, and Shorvon[31] gave 125 to 150 mg. daily for more than four years. Compared with the daily amount of amphetamine administered over long periods by the authors listed, the amphetamine content of Lakobie capsules, varying from 5 to 15 mg. in the different formulas, does not seem excessive, and a recommended daily dose of 15 to 45 mg. of amphetamine hydrochloride appears to be well within safe limits.

Thyroid Content: The opinions of authors are not uniform with respect to the use of thyroid in the treatment of obesity. While some,[32, 33, 34] conclude that thyroid should be employed only when the diagnosis of hypothyroidism has been definitely established, others[35] state that in cases of uncomplicated obesity, when diet alone does not produce the desired results, thyroid can be employed safely, provided that the patient remains under close medical supervision. The average dose given by Bayer and Gray[35] was 1½ grains (about 100 mg.) of thyroid daily. Bethea[36] stated, "In obesity, thyroid has a field of usefulness even in the presence of active mentality and normal basal metabolic reading." In doubtful cases the best indication for thyroid therapy is the symptomatic response to thyroid medication.[37, 12, 13] Goldzieher[38] administered thyroid in obesity due to a pituitary disorder in addition to pituitary preparations.

Phenobarbital Content: The addition of phenobarbital to the Lakobie formulas serves a dual purpose: it counteracts any overstimulating effect due to the combined action of amphetamine hydrochloride and thyroid, and it minimizes or prevents insomnia. It may be added that with protracted medication with amphetamine the euphoria and waking effects decrease, because of the cumulative need for rest.

Although the constipating effects of prolonged amphetamine medication were kept in mind, a laxative was not included in the Lakobie formulas. This deliberate omission was based on the fact that individual costive reactions to amphetamines vary considerably and can be minimized by a properly selected bulky diet. Furthermore, the need for a laxative and its dosage should be left to the judgment of the attending physician.

Studies and Results of Treatment with Lakobie

Thus far, 100 unselected obese patients have been treated with Lakobie formulas, 65 of them with the most potent formula, Lakobie 15, twenty with Lakobie 10 and fifteen with Lakobie 5. In all cases the loss of weight, the blood pressure and the pulse rate have been checked carefully and the results recorded. The ages of the patients varied from 15 to 67 years and their weights from 142 pounds (64 Kg.) to 287½ pounds (130 Kg.). Weight reduction was obtained in all cases, the degree depending on the individual reaction to the drug and cooperation with respect to diet. Weight losses ranged from 10 pounds (4.5 Kg.) to 50 pounds (23 Kg.). Treatment time was extended in some cases to more than nine months. The initial loss of weight in some cases was rather dramatic; some patients lost as much as 10 pounds the first week, 1 patient lost 26 pounds (12 Kg.) in two weeks, another lost 26 pounds in five weeks and a third lost 26 pounds in ten weeks. It took one patient three months to lose 34 pounds (15 Kg.). The patient who lost 50 pounds was treated for nine months. Generally the attempted average weekly reduction was 2½ to 3 pounds (about 1 to 1.5 Kg.).

The blood pressure in the majority of the treated patients was normal at the commencement of the reducing therapy, while in a number of cases systolic elevations of blood pressure to 180 were observed and one patient

had a reading of 200 systolic and 140 diastolic before treatment was started. (Riva-Rocci's sphygmomanometer was used in all tests.) In this latter case the reaction to the drug was studied with special care; the patient's blood pressure was reduced to 160 systolic and 100 diastolic within three weeks, during which time her weight decreased 15 pounds (7 Kg.). The reduction of blood pressure in slightly hypertensive patients during treatment was a common occurrence. Elevations in blood pressure during treatment were rare and transient. Elevations of 10 to 20 mm. of mercury were observed in some cases; however, the blood pressure returned to normal or close to normal during continued medication. One patient showed an elevation of blood pressure from 160 systolic and 100 diastolic to 200 systolic and 130 diastolic about three hours after she had, in error, taken 3 capsules of Lakobie 15 (45 mg. of amphetamine hydrochloride) in a single dose. Her blood pressure was checked the two following days and found to be 150 systolic and 90 diastolic; treatment had continued during this period.

The pulse rate in the majority of the patients remained normal, varying from 70 to 80 per minute. In some cases the pulse rate increased to 85 to 90 per minute and remained at this level for some time. Increases in pulse rate to around 100 were rare, and pronounced tachycardia was not observed in any case.

Undesirable side effects during treatment with Lakobie formulas were few. The most commonly encountered side effect was dryness of the mouth, which the patient could counteract by chewing gum. Constipation occurred in a number of cases and was corrected by diet and with laxatives (Glysennid, cascara or aloin). Some patients complained of slight dizziness and mild insomnia; these symptoms became milder and finally disappeared with continued medication. Medication was discontinued in only 2 patients; one was a patient who exhibited extreme hyperexcitability, and the other was hypersensitive to amphetamine, with resultant dizziness.

The amount of Lakobie prescribed per day in no case exceeded 3 capsules of the most potent formula (45 mg. of amphetamine hydrochloride). At the beginning of the study, 1 capsule was generally given one hour before breakfast and one hour before lunch; the third capsule was given not later than 4 p. m., to prevent interference with sleep. In the course of the investigation the eating habits of the patients were studied closely, and it was found that most patients had no difficulty in limiting or eliminating completely the caloric intake at breakfast. Therefore, the medication before breakfast was omitted in those cases. The dosage was then adjusted to obtain the highest anorexigenic effect at the time when the most intense hunger sensation usually occurred. It was found that many patients reacted satisfactorily when Lakobie was given one hour before lunch and at 4 p. m. In cases in which some tolerance to Lakobie developed, treatment was interrupted for ten to fourteen days, after which medication was resumed with good results.

Summary and Conclusions

1. Obesity is caused by overeating in the vast majority of cases.

2. Obesity is a danger to health and longevity.

3. Efficient reduction in weight cannot be achieved without restrictive diet.

4. Anorexigenic compounds have a definite place in the treatment of obesity in conjunction with reducing diets.

5. Lakobie formulas have been thoroughly investigated and have been found valuable and safe adjuncts to restrictive diets in the treatment of obesity.

REFERENCES

1. Carey, L. S.: *M. Clin. North America* 23:1449, Nov. 1939.
2. McLester, J. S.: *Nutrition and Diet in Health and Disease*, ed. 4, W. B. Saunders Company, 1943, pp. 432–459.
3. Danowski, T. S. and Winkler, A. W.: *Am. J. M. Sc.* 208:622, Nov. 1944.
4. Preble, W. E.: *Boston M. & Surg. J.* 188:617, April 26, 1923.
5. Beardwood, J. T., Jr.: *Obesity, Cyclopedia of Medicine, Surgery and Specialties*, ed. 2, Philadelphia, F. A. Davis Company, 1945, vol. 10, pp. 706-739.
6. Newburgh, L. H.: Obesity, *Arch. Int. Med.* 70:1033, Dec. 1942; *Physiol. Rev.* 24:18, Jan. 1944.
7. Evans, F. A.: *Obesity in Duncan, G. G.: Diseases of Metabolism*, Philadelphia, W. B. Saunders Company, 1942; *Penn. M. J.* 42:1174, July 1939
8. Bruch, H.: *Am. J. Psychiat.* 99:752, March 1943.
9. Bram, I.: *M. Rec.* 157:673, Nov. 1944.
10. Rennie, T. A. C.: *Dis. Nerv. System* 1:238, Aug. 1940.
11. Gurney R.: The Heredity Factor in Obesity *Arch. Int. Med.* 57:557 March 1943.
12. Barr, D. B.: *Obesity, Modern Medical Therapy in General Practice*, Baltimore, Williams & Wilkins Company, 1940, vol. 1, pp. 922–944.
13. Wohl, M. G.: *Dietotherapy*, Philadelphia, W. B. Saunders Company, pp. 791–813, 1945.
14. Bauer, J.: Obesity: Its Pathogenesis, Etiology and Treatment, *Arch. Int. Med.* 67:968, May 1941.
15. Baborka, C. J.: *M. Clin. North America* 21:23, Jan. 1937.
16. *Am. J. Roentgenol.* 36:868, 1936.
17. Beyer, K. H.: *J. Pharmacol. & Exper. Therap.* 66:318, 1939.
18. Bram, I.: *Internat. Clin.* 1:48, 1937.
19. Rehfuss, M. E., Albrecht, F. K. and Price, A. H.: *A Course in Practical Therapeutics*, Baltimore, Williams & Wilkins Company, 1948.
20. *New and Nonofficial Remedies*, Philadelphia, J. B. Lippincott Company, 1948.
21. Harper: *North Carolina M. J.* 6:96–98, Feb. 1945.
22. Myerson: *Am. J. M. Sc.* 199:729, May 1940.
23. Mathews: *Am. J. M. Sc.* 195:448, 1938.
24. Hahne, L. S.: Addiction to Amphetamine Sulfate, *J. A. M. A.* 115:1568, Nov. 1940.
25. Bakst: *U. S. Nav. M. Bull.* 43:1228, Dec. 1944.
26. Ulrich, H.: *New Engl. and J. Med.* 217:696, Oct. 28, 1937.
27. Myerson, A. and Lonano, J. L.: *Arch. Neurol. & Psychiat.* 48:823, Nov. 1942.
28. Bloomberg: *New Engl. and J. Med.* 222:946, June 6, 1940.
29. Gorrell: *Clin. M. Surg.* 45:318, July 1938.
30. Korns, R.: *Am. Heart J.* 13:144, 1937; *Ann. Int. Med.* 12:233, Aug. 1938.
31. Shorvon, H. J.: *Brit. M. J.* 2:285, Sept. 1945.
32. Von Noorden, C.: *Wien. med. Wschr.* 81:23, 1931.
33. Means, J. H.: *The Thyroid and Its Diseases*, Philadelphia, J. B. Lippincott Company, 1937.
34. Strang, J. M., McClugage, H. B. and Evans, F. A.: *J. A. M. A.* 97:1063, Oct. 10, 1931.
35. Bayer, L. M. and Gray, H.: *Am. J. M. Sc.* 189:86, 1935.
36. Bethea, O. W.: *Intat. M. Digest* 29:371, Dec. 1936.
37. Rose, E.: *M. Clin. North America* 26:1711, Nov. 1942.
38. Goldzieher, M. A.: *Practical Endocrinology*, ed. 2, New York, D. Appleton-Century Company, Inc., 1937.

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The Alcoholic Woman

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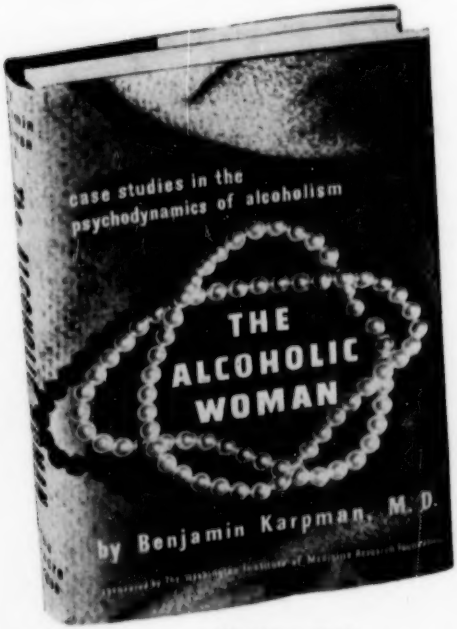

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